Editorial

Commotio cordis: sudden death due to chest wall impact in sports

Sudden death resulting from relatively minor chest wall blows (commotio cordis) has been described in the medical literature since the late 1970s.[1] Since this phenomenon was first described, 70 cases have been collected (Barry J Maron, personal communication, 1998). This number is, in all likelihood, an underestimate of the true incidence of this phenomenon as many cases go unreported or are reported as accidental deaths or idiopathic sudden death. Indeed, a case of ventricular fibrillation during a soccer match, although initially reported as idiopathic, was on careful review of the events, discovered to occur after an elbow blow to the anterior left chest[2] (Gianfranco Buja, personal communication, 1998). Commotio cordis occurs most frequently in baseball, but sudden death owing to chest impact has also been reported in hockey, lacrosse, softball, and after bodily impacts in other sports.[1–6] There are no known cases in cricket, but given that the ball is quite similar in size, weight, and hardness to a baseball, cricket players may also be at risk for commotio cordis. The lack of deaths resulting from chest wall impact in cricket matches is likely because of the lower numbers of individuals playing the game (compared to baseball) and to the frequency of batsmen wearing protective chest gear. However, it is also possible that deaths resulting from chest impact in cricket may not be reported as commotio cordis as there is a relative lack of awareness of this event.

Human data

Human victims of commotio cordis are young, healthy, and predominantly male.[3] The victims have no known histories of heart disease or other chronic medical illnesses. The impacts occur on the chest wall directly overlying the heart, and the velocity of the impact is routine for the sport involved.[3] Collapse is usually instantaneous, but occasional brief moments of lucidity in which the victim complains of lightheadedness have also been described. Ventricular fibrillation is the most common initial rhythm documented; however, heart block, accelerated idioventricular rhythm, and asystole have also been seen.[1–7] Resuscitation is more difficult than would be expected, especially as the athletes do not have cardiac disease and there are often immediate resuscitative efforts.[3 8] Necropsy reveals no thoracic or cardiac abnormalities. In these unfortunate victims, the most widely believed mechanism of sudden death is ventricular fibrillation resulting from impact during a vulnerable period of the cardiac cycle.[3] Other proposed causes are complete heart block, a pronounced vagal response, and electromechanical dissociation.[9]

Experimental models

Previous experimental models of chest wall impact used high energy impacts that almost universally caused severe thoracic and cardiac damage.[10 11] These models, therefore, were more reflective of motor vehicular accidents and bomb blasts than commotio cordis. In an effort to delineate the mechanism of sudden death in low energy chest wall impacts, we recently developed an experimental model of low energy chest wall impact.[12] In this model, a 30 mph baseball impact was given to the chest wall of anaesthetised juvenile pigs. The impact was gated to the cardiac cycle. With the use of an electrophysiological stimulator to control the release of the impact object and a known 130 ms flight time, the impact could be delivered at any chosen time during the cardiac cycle. In this model, ventricular fibrillation was reproducibly initiated by impacts during a 15 ms time window on the upslope of the T wave (30 to 15 ms before the peak of the T wave). The initiation of ventricular fibrillation was instantaneous with the impact and was not preceded by ischaemic ST segment changes, extrasystoles, bradyarrhythmias, or ventricular tachycardias. Ventricular fibrillation was not produced with impacts during other parts of the cardiac cycle. Short bursts of polymorphic ventricular tachycardia were seen with impacts just outside the vulnerable zone for ventricular fibrillation. In addition, transient complete heart block (up to seven beats) was produced with QRS segment impacts, and left bundle branch blocks and ST segment elevation were produced by impacts throughout the cardiac cycle. Immediate angiography did not reveal epicardial coronary abnormalities. Transient apical wall motion abnormalities and mild apical perfusion defects were occasionally observed. No animal had thoracic pathology or major cardiac abnormalities. The findings in this model simulated the human findings in commotio cordis.

In further experiments with this model, we demonstrated that the hardness of the impact object directly correlated with the risk of inducing ventricular fibrillation with impact.[12] With a very soft impact object (Reduced Injury Factor (RIF) 1 baseball; Worth Inc, Tullahoma, Tennessee, USA) ventricular fibrillation was induced in 8% of the impacts in the vulnerable time zone. With a standard baseball, ventricular fibrillation was produced in 35% of the impacts, and with a wooden block, ventricular fibrillation was induced in 90% of the impacts. Cricket balls have a hardness that is similar to a regulation baseball. Therefore, an appropriately timed cricket impact directly over the cardiac silhouette could theoretically result in sudden death.

Conclusion

Commotio cordis—sudden death resulting from low energy chest wall impact—although rare, may be more common that previously believed. In our animal model, ventricular fibrillation was reproducibly produced with a 30 mph impact during a 15 ms window on the upslope of the T wave, and the hardness of the impact object directly correlated with the risk of sudden death. Athletes in many sports are at risk (albeit small). Efforts at decreasing the risks are already in practice (in the United States, baseball leagues for young athletes are increasingly using "safety" baseballs). Other preventive methods such as chest wall protectors may also be useful. Further research is needed to define more effective preventive and treatment measures for this devastating condition.

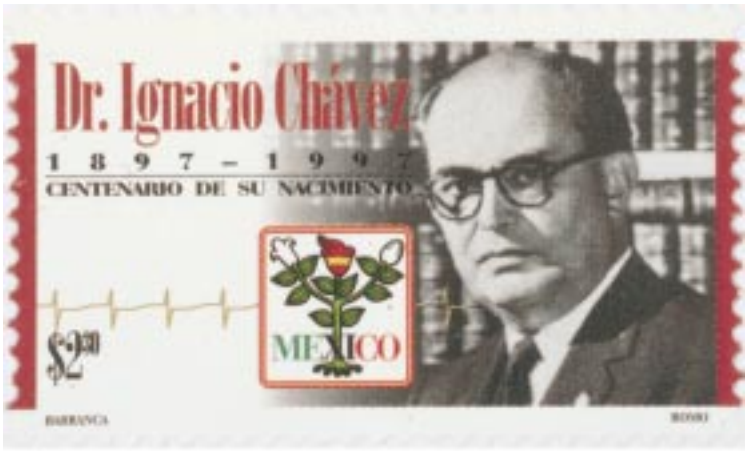
MARK S LINK

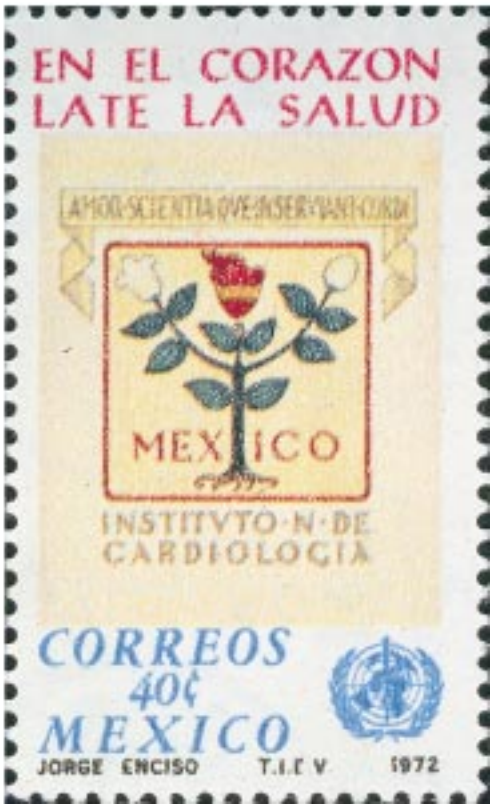
Cardiac Arrhythmia Service,
New England Medical Center, Boston, MA 02111, USA

1 Dickman GL, Hassan A, Luckstead EF. Ventricular fibrillation following baseball injury. *Physician and Sport Medicine* 1978;**6**:85–6.
2 Buja G, Meneghello MP, Bellotto F. Isolated episode of exercise-related ventricular fibrillation in a healthy athlete. *Int J Cardiol* 1989;**24**:121–3.
3 Maron BJ, Poliac LC, Kaplan JA *et al*. Blunt impact to the chest leading to sudden death from cardiac arrest during sports activities. *N Engl J Med* 1995;**333**:337–42.
4 Kaplan JA, Karofsky PS, Volturo GA. Commotio cordis in two amateur ice hockey players despite the use of commercial chest protractors: case reports. *J Trauma* 1993;**34**:151–3.
5 Abrunzo TJ. Commotio cordis, the single, most common cause of traumatic death in youth baseball. *Am J Dis Child* 1991;**145**:1279–82.
6 Link MS, Ginsburg SH, Wang PJ, *et al*. Commotio cordis: cardiovascular manifestations of a rare survivor. *Chest* 1998;**114**:326–8.
7 Maron BJ, Strasburger JF, Kugler JD, *et al*. Survival following a blunt chest impact induced cardiac arrest during sports activities in young athletes. *Am J Cardiol* 1997;**79**:840–1.
8 Link MS, Wang PJ, Pandian NG, *et al*. Resuscitation in a biological model of commotio cordis, sudden death from low energy chest wall impact [abstract]. *J Am Coll Cardiol* 1998;**31**:403A.
9 Estes NAM III. Sudden death in young athletes. *N Engl J Med* 1995;**333**:380–1.
10 Cooper GJ, Pearce BP, Stainer MC, *et al*. The biomechanical response of the thorax to nonpenetrating impact with particular reference to cardiac injuries. *J Trauma* 1982;**22**:994–1008.
11 Viano DC, Andrzejak DV, Polley TZ, *et al*. Mechanism of fatal chest injury by baseball impact: development of an experimental model. *Clin J Sport Med* 1992;**2**:166–71.
12 Link MS, Wang PJ, Pandian NG, *et al*. An experimental model of sudden death due to low energy chest wall impact (commotio cordis). *N Engl J Med* 1998;**338**:1805–11.

STAMPS IN CARDIOLOGY

Instituto Nacional de Cardiologia Ignacio Chavez





Mexico issued a stamp in 1997 to commemorate the centenary of the birth of Dr Ignacio Chavez. In addition to showing his portrait the stamp incorporates the ECG and the emblem of the National Institute of Cardiology of Mexico. This emblem is also the design feature of the 1972 stamp from Mexico issued to mark the World Health Month of the World Health Organisation, the theme for that year being "Your heart is your health". This stamp was designed by J Enciso and two million were issued in sheets of 50. The stamp comes from a set of two—the second stamp featuring Willem Einthoven appeared in *Heart* 1997;**78**:324.

It was the foresight and dedication of Dr Ignacio Chavez (1897–1979) that led to the National Institute of Cardiology of Mexico being founded in 1944 when it was the first such institute in the world. Dr Chavez had the inspiration of presenting the history of cardiology in the entrance hall of the building by means of a series of large mural paintings. These were created by the renowned Mexican artist Diego Rivera and they are justly famous for their vivid illustrations of cardiology from the time of Galen onwards. The emblem of the institute is a Mexican plant called *Yoloxochitl*, the heart flower, which was used before the time of Columbus to treat dropsy. It has a weak digitalis-like action. The scientific name is *Talauma mexicana*, a member of the magnolia family. The emblem was designed by Diego Rivera and it is taken from an illustration in the Codex Badianus of 1552, which was the first book on therapeutics in the New World. It shows the plant with an Aztec stylised heart above it. We are most grateful to Dr Ignacio Chavez-Rivera, the director of the Institute, for his invaluable assistance.

M K DAVIES
A HOLLMAN